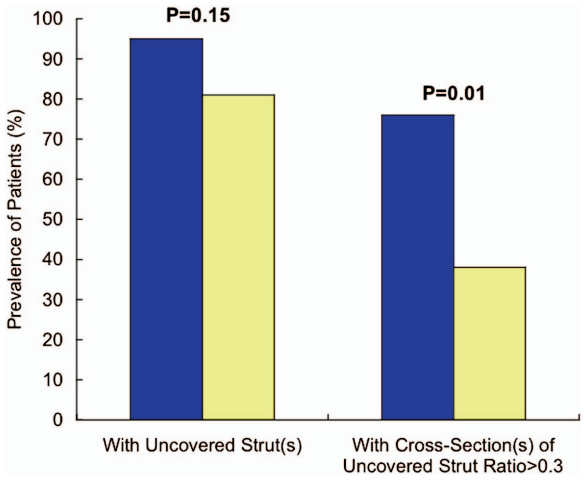

Figure 2 **Prevalence of Patients With Uncovered Struts**

The prevalence of patients with uncovered strut(s) did not differ between the 3-month follow-up group **(blue bars)** and the 2-year follow-up group **(yellow bars)**. In contrast, prevalence of patients with cross-section(s) of uncovered strut ratio >0.3 was lower in the 2-year group than in the 3-month group.

cross-sections, additional special factors, such as discontinuation of antiplatelet therapy, antiplatelet resistance, intrinsic thrombogenicity, or coronary flow problems might trigger the development of LST.

In comparison with previous data of 3-month follow-up (patients n = 21, struts n = 4,545) (3), thickness of NIH in the 2-year follow-up group was greater than that in the 3-month follow-up group (71 ± 93 μm vs. 29 ± 41 μm, respectively; $p < 0.00001$). Frequency of uncovered struts was lower in the 2-year group than in the 3-month group (5% vs. 15%, respectively; $p < 0.00001$). Moreover, the prevalence of patients with cross-section(s) of uncovered strut ratio >0.3 was lower in the 2-year group than in the 3-month group (38% vs. 76%, respectively) (Fig. 2). Clinical characteristics were similar between the 2 groups. From this perspective, neointimal coverage inside the SES progressed, and uncovered stent struts decreased from 3-month to 2-year follow-up. In contrast, prevalence of patients with uncovered struts did not differ between the 3-month group and the 2-year group (95% vs. 81%, respectively) (Fig. 2). Despite the fact that neointimal coverage advanced, a few troublesome stent struts might persist as uncovered struts for up to 2 years.

Two thrombi inside the SES were identified in patients who did not have an uncovered strut. Cross-sectional OCT images might have a limitation in identification of the uncovered struts under in-stent thrombi. Intracoronary structures inside the SES, except for distinct thrombi, were regarded as NIH in this OCT analysis. However, it was possible that some tissue of fluffy appearance (Fig. 1) was not NIH but was actually fibrin deposition. Unfortunately, no method of distinguishing fibrin layer from NIH around the struts in living patients has been established. Additionally, there is no confirmation that morphological intracoronary structures have mature endothelial function, such as an antithrombotic function. Although OCT is used, evaluation for the uncovered struts might be underestimated in the situation of fibrin deposition or thrombus adhesion to the struts.

In conclusion, long-term follow-up OCT examination demonstrated that few uncovered struts remained in the majority of the patients for up to 2 years after SES implantation. Further careful follow-up might be required.

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Letters to the Editor

T-Wave Alternans and Intraventricular Conduction Delays

I read with great interest the study by Cantillon et al. (1) on the utility of microvolt T-wave alternans (MTWA) in predicting total mortality and arrhythmia-free survival in patients with a left ventricular ejection fraction ≤30% who had been referred for invasive electrophysiological testing for evaluation of syncope and/or nonsustained ventricular tachycardia. It is not clear to this reader whether this report's database derives from the one employed in another study, emanating from the same laboratory, and published in another journal on the same month as the present study (2). However, even if this is the case, the present study provides an opportunity to evaluate the influence of the QRS duration on the magnitude of the MTWA. The authors in both studies (1,2) employed the traditional threshold of ≥1.9 μV, derived from the spectral analysis of the signal, to characterize an

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MTWA value as positive. Although this analysis operates on this dichotomy to generate the "positive" and the "negative" response of a patient to MTWA testing, thus creating a qualitative context, there is a quantitative issue as its underpinning (i.e., a patient has to reach the threshold to qualify as "positive"). Moreover, current work indicates that the magnitude in μV of MTWA may be of importance, and higher values of calculated MTWA may imply worse prognosis (3,4), or therapy with various drugs, particularly beta-blockers, may attenuate the magnitude of MTWA (5). However, more work is needed to substantiate the claim that the quantitative assessment of MTWA may have advantages over the qualitative evaluation. In the meantime, it is important to deal with some possible confounders of the quantitative employment of the MTWA, 1 of which may be its T-wave amplitude dependence (6). Accordingly, in the presence of ventricular conduction delays, which often are associated with T waves larger in amplitude than the ones encountered in association with normal intraventricular conduction, a greater magnitude of MTWA in the former than in the latter may not mean a higher risk for sudden cardiac death or malignant ventricular arrhythmia, but merely an enhancing effect of the taller T waves on the magnitude of MTWA in patients with wide QRS complexes. Probably this was the reason for the increased rate of false-positive MTWA tests in patients with wide QRS complexes (≥120 ms) (specificity 22% in the patients with a wide QRS and 40% in the patients with a narrow QRS) in the authors' other report (2). In that report they categorized their patients to those with QRS duration of <120 ms and ≥120 ms (2). In their present contribution (1), by employing 3 categories of patients in terms of QRS duration (Table 1) (QRS <110 ms, QRS 110 ms to 120 ms, and QRS >120 ms), they provide an opportunity to evaluate the effect of the amplitude of the T waves on the magnitude of MTWA in a "dose response" fashion. What is needed for this is a comparison of the values of MTWA in μV, and the T-wave amplitudes in mV in the above 3 categories of patients, according to their QRS duration. Could the authors provide us with these data? Evaluation of possible determinants of the MTWA and potential adjustment via a MTWA index (6) may upgrade both the currently used "qualitative" assessment and a future adoption of quantitative assessment of MTWA testing.

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Percutaneous Intervention in Saphenous Vein Bypass Graft Disease

Case Against the Use of Drug-Eluting Stents

Saphenous vein grafts (SVGs) tend to degenerate over time, and almost one-half develop significant stenosis and nearly 40% are completely occluded within 1 decade (1). Use of balloon angioplasty alone without stenting for treatment of SVG disease is associated with poor short-term and long-term outcome (2). Use of bare-metal stents (BMS) is associated with restenosis rates as high as 50% at 6 months (3). Lately, drug-eluting stents (DES) have been used for the treatment of SVG disease.

In contrast to the native coronary arteries, in which restenosis after percutaneous coronary intervention (PCI) is mainly due to intimal hyperplasia, restenosis in SVG involves cellular hyperplasia, progression of atherosclerosis, local inflammatory reaction to stent struts, and thrombosis. These observations suggest that it may not be wise to extend the seemingly better short- and midterm results seen with PCI for treatment of native coronary artery disease to the treatment of SVG disease. We have recently looked at the data in our institution in 109 patients with SVG disease who received BMS or DES. During a follow-up period of 33 months, we found that the incidence of

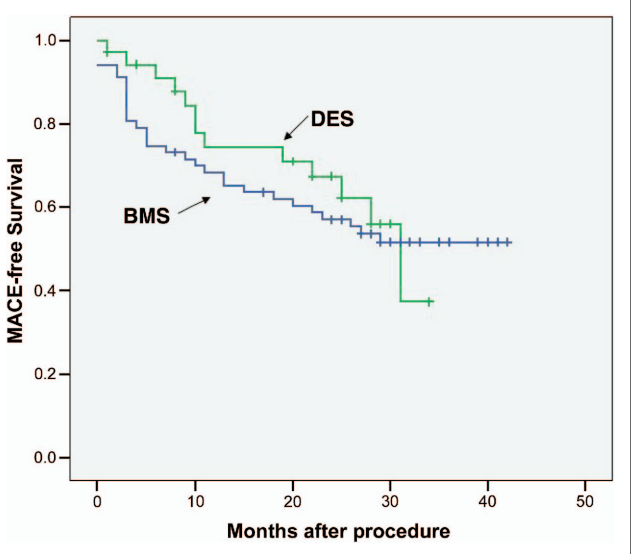

Figure 1 **Kaplan-Meier Survival Curves for Freedom From MACE at Almost 3 Years Follow-Up**

BMS = bare-metal stents; DES = drug-eluting stents; MACE = major adverse cardiac events.